

**Listing of Claims:**

Please delete the claims in the specification and replace them with the following new claims.

Claims 1-31 cancelled.

Claim 32. (NEW) A process for the in vitro differentiation of neuronal stem cells and of cells derived from neuronal stem cells, comprising

- (a) contacting the cells with a substance which inhibits a reaction of the Wnt signal transduction pathway, and
- (b) culturing said cells under conditions which enable said cells to propagate and/or differentiate.

Claim 33. (NEW) The process as claimed in claim 32, characterized in that the cells differentiate into brain cell-like cells.

Claim 34. (NEW) The process as claimed in claim 32, characterized in that, where appropriate, a step (c) comprises determining the concentration of a protein of the Wnt signal transduction pathway.

Claim 35. (NEW) The process as claimed in claim 34, characterized in that the protein concentration is determined by means of an antibody.

Claim 36. (NEW) The process as claimed in claim 35, characterized in that the protein is  $\beta$ -catenin.

Claim 37. (NEW) The process as claimed in claim 32 characterized in that the reaction of the Wnt signal transduction pathway is inhibited by way of inhibition of glycogen synthase kinase 3.

Claim 38. (NEW) The process as claimed in claim 37, characterized in that glycogen synthase kinase 3 is inhibited by at least one inhibitor selected from the group consisting of kinase inhibitors, estrogen analogs, phytoestrogens, corticoids and salts, in particular 4-benzyl-2-methyl-1,2,4-thiazolidine-3,5-dione, 2-thio(3-iodobenzyl)-5-(1-pyridyl)-[1,3,4]-oxadiazole, 3-(2,4-dichlorophenyl)-4-(1-methyl-1H-indol-3-yl)-1H-pyrrole-2,5-dione, 3-[(3-

chloro-4-hydroxyphenyl)amino]-4-(2-nitrophenyl)-1H-pyrrole-2,5-dione, lithium salts, beryllium salts.

Claim 39. (NEW) The process as claimed in claim 38, characterized in that the inhibitor is genistein.

Claim 40. (NEW) The process as claimed in claim 39, characterized in that genistein is used in a concentration of 10-250  $\mu\text{mol/l}$ .

Claim 41. (NEW) The process as claimed in claim 32, characterized in that the reaction of the Wnt signal transduction pathway is inhibited by at least one antagonist of the Frizzled receptor.

Claim 42. (NEW) The process as claimed in claim 41, characterized in that the at least one antagonist is selected from the group consisting of secreted Frizzle-related proteins (sFRP), Dickkopf (Dkk), Wnt, Fzd, Frat, Nkd, VANG1/STB2, ARHU/WRCH1, ARHV/WRCH2, GIPC2, GIPC3, betaTRCP2/FBXW1B, SOX17, TCF-3, WIF-1, Cerberus, Sizzled, Crescent, Coco, Soggy, Kremen and low-density-lipoprotein-receptor-related proteins (LRP).

Claim 43. (NEW) The process as claimed in claim 32, characterized in that the cells derived from neuronal stem cells are cells selected from the group consisting of neuroblastoma cells, PC12 cells, cells of neuronal primary cultures and 293 cells.

Claim 44. (NEW) A cell obtainable by the process as claimed in claim 32.

Claim 45. (NEW) A neurological tissue replacement comprising cells as claimed in claim 44.

Claim 46. (NEW) A pharmaceutical agent comprising cells as claimed in claim 44.

Claim 47. (NEW) A screening process for identifying substances which inhibit the Wnt signal transduction pathway and are suitable for the differentiation of neuronal stem cells and of cells derived from neuronal stem cells, comprising

- (c) contacting said cells with said substance,
- (d) determining the  $\beta$ -catenin concentration in said cells,

- (e) comparison with a suitable comparative cell, and
- (f) detecting differentiation of said cells.

Claim 48. (NEW) A pharmaceutical agent comprising inhibitors of glycogen synthase kinase 3, antagonists of the Frizzled receptor and/or antibodies to proteins of the Wnt signal transduction pathway.

Claim 49. (NEW) The agent as claimed in claim 48, characterized in that the inhibitor of glycogen synthase kinase 3 is genistein.

Claim 50. (NEW) The method of treating a disease on which modulation of the activity or amount of a protein of the Wnt signal transduction pathway may have a beneficial influence comprising administering a pharmaceutical agent as claimed in claim 46 in an amount sufficient to treat the disease.

Claim 51. (NEW) The method of treating a disease on which modulation of the activity or amount of a protein of the Wnt signal transduction pathway may have a beneficial influence comprising administering a pharmaceutical agent as claimed in claim 48 in an amount sufficient to treat the disease.

Claim 52. (NEW) The method of treating a disease on which modulation of the activity or amount of a protein of the Wnt signal transduction pathway may have a beneficial influence comprising administering a pharmaceutical agent as claimed in claim 49 in an amount sufficient to treat the disease.

Claim 53. (NEW) The method of treating as claimed in claim 50, characterized in that the disease is one selected from the following groups:

the group of cerebral malformations, in particular of cerebral developmental anomalies, cerebral palsies in infants, craniocervical junction abnormalities and dysraphic syndromes,

the group of degenerative and atrophic processes of the brain and the spinal cord, in particular of senile and presenile atrophies of the brain, in particular Alzheimer's disease, Binswanger's disease and Pick's disease,

the group of basal ganglia disorders, in particular Huntington's disease and HDL2, chorea, athetosis and dystonia, spongiform encephalopathies,

the group of degenerations of the corticospinal tract and of the anterior horn of the spinal cord, in particular amyotrophic lateral sclerosis, spinal muscular atrophy and progressive bulbar paralysis,

the group of degenerative ataxias, in particular Friedreich's disease, Refsum's disease and spinocerebellar ataxias type 1-25,

the group of metabolic and toxic processes of the brain and of the spinal cord, Wilson's disease, multiple sclerosis, demyelinating diseases of the central and peripheral nerve system, brain and spinal cord tumors, traumatic damage to the nerve system, circulation disorders of the brain and the spinal cord, in particular hereditary metabolic disorders of the amino acid, lipid, carbohydrate and metal ion metabolisms, in particular Wilson's disease, multiple sclerosis, cerebral infarctions and other forms of stroke, muscular disorders based on damage to the nerve system and post-traumatic muscular atrophies.

Claim 54. (NEW) The method of treating as claimed in claim 51, characterized in that the disease is one selected from the following groups:

the group of cerebral malformations, in particular of cerebral developmental anomalies, cerebral palsies in infants, craniocervical junction abnormalities and dysraphic syndromes,

the group of degenerative and atrophic processes of the brain and the spinal cord, in particular of senile and presenile atrophies of the brain, in particular Alzheimer's disease, Binswanger's disease and Pick's disease,

the group of basal ganglia disorders, in particular Huntington's disease and HDL2, chorea, athetosis and dystonia, spongiform encephalopathies,

the group of degenerations of the corticospinal tract and of the anterior horn of the spinal cord, in particular amyotrophic lateral sclerosis, spinal muscular atrophy and progressive bulbar paralysis,

the group of degenerative ataxias, in particular Friedreich's disease, Refsum's disease and spinocerebellar ataxias type 1-25,

the group of metabolic and toxic processes of the brain and of the spinal cord, Wilson's disease, multiple sclerosis, demyelinating diseases of the central and peripheral nerve system, brain and spinal cord tumors, traumatic damage to the nerve system, circulation disorders of the brain and the spinal cord, in particular hereditary metabolic disorders of the amino acid, lipid, carbohydrate and metal ion metabolisms, in particular Wilson's disease, multiple sclerosis, cerebral infarctions and other forms of stroke, muscular disorders based on damage to the nerve system and post-traumatic muscular atrophies.

Claim 55. (NEW) The method of treating as claimed in claim 52, characterized in that the disease is one selected from the following groups:

the group of cerebral malformations, in particular of cerebral developmental anomalies, cerebral palsies in infants, craniocervical junction abnormalities and dysraphic syndromes,

the group of degenerative and atrophic processes of the brain and the spinal cord, in particular of senile and presenile atrophies of the brain, in particular Alzheimer's disease, Binswanger's disease and Pick's disease,

the group of basal ganglia disorders, in particular Huntington's disease and HDL2, chorea, athetosis and dystonia, spongiform encephalopathies,

the group of degenerations of the corticospinal tract and of the anterior horn of the spinal cord, in particular amyotrophic lateral sclerosis, spinal muscular atrophy and progressive bulbar paralysis,

the group of degenerative ataxias, in particular Friedreich's disease, Refsum's disease and spinocerebellar ataxias type 1-25,

the group of metabolic and toxic processes of the brain and of the spinal cord, Wilson's disease, multiple sclerosis, demyelinating diseases of the central and peripheral nerve system, brain and spinal cord tumors, traumatic damage to the nerve system, circulation disorders of the brain and the spinal cord, in particular hereditary

metabolic disorders of the amino acid, lipid, carbohydrate and metal ion metabolisms, in particular Wilson's disease, multiple sclerosis, cerebral infarctions and other forms of stroke, muscular disorders based on damage to the nerve system and post-traumatic muscular atrophies.

Claim 56. (NEW) The method of treating a disease which lead directly or indirectly to the death of brain cells, comprising administering genistein in an amount sufficient to treat the disease.

Claim 57. (NEW) A screening process for detecting brain cell-like cells and brain cells, comprising

- (i) determining the concentration of  $\beta$ -catenin, and
- (ii) comparing the concentration from (i) with the  $\beta$ -catenin concentration of a suitable comparative cell.

Claim 58. (NEW) The screening process as claimed in claim 57, characterized in that the  $\beta$ -catenin concentration is determined by means of an antibody.

Claim 59. (NEW) The method of use of  $\beta$ -catenin as diagnostic marker for identifying brain cell-like cells and brain cells.

Claim 60. (NEW) An in vitro differentiation of recombinant, neuronal stem cells into brain cell-like cells, effected by a nucleic acid construct for expressing a protein capable of inhibiting a reaction of the Wnt signal transduction pathway.

Claim 61. (NEW) The differentiation as claimed in claim 60, characterized in that the protein is expressed under the control of a constitutive or of a regulatable promoter.

Claim 62. (NEW) The differentiation as claimed in claim 60, characterized in that the cell has been transfected stably or transiently with the nucleic acid construct.

Claim 63. (NEW) A differentiation of a recombinant, neuronal stem cell into brain cell-like cells, effected by at least one protein of the Wnt signal transduction pathway not being expressed, being expressed inactively or being expressed at a reduced level in comparison with the corresponding wild type stem cell.

Claim 64. (NEW) The differentiation as claimed in claim 63, characterized in that at least one gene coding for a protein of the Wnt signal transduction pathway or a DNA section involved in expression of said gene has been completely or partially deleted or has a mutation.

Claim 65. (NEW) A kit for in vitro differentiation of neuronal stem cells and of cells derived from neuronal stem cells, comprising a recombinant, neuronal stem cell which comprises a nucleic acid construct for expressing a protein capable of inhibiting a reaction of the Wnt signal transduction pathway.

Claim 66. (NEW) A kit for in vitro differentiation of neuronal stem cells and of cells derived from neuronal stem cells, comprising a recombinant, neuronal stem cell in which at least one protein of the Wnt signal transduction pathway is not expressed, is expressed inactively or is expressed at a reduced level in comparison with the corresponding wild type stem cell.